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10/009,861	07/03/2002	Carlos Cordon-Cardo	55293-B-PCT-US/JPW/FHB	6709

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EXAMINER

UNGAR, SUSAN NMN

ART UNIT PAPER NUMBER

1642

DATE MAILED: 02/03/2005

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary

Application No.

10/009,861

Applicant(s)

CORDON-CARDO ET AL.

Examiner

Susan Ungar

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-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☐ Responsive to communication(s) filed on 06 August 2004.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☐ Claim(s) 1,2,4-6,9-16,18,19 and 25-30 is/are pending in the application.
- 4a) Of the above claim(s) 1-2, 4-6, 9-16, 18, 25-26 and 30 is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☐ Claim(s) 10 and 27-29 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some * c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
 2. ☐ Certified copies of the priority documents have been received in Application No. _____.
 3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- 1) ☒ Notice of References Cited (PTO-892)
- 2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 3) ☒ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08)
Paper No(s)/Mail Date 3/6/03, 10/15/02.
- 4) ☐ Interview Summary (PTO-413)
Paper No(s)/Mail Date. _____.
- 5) ☐ Notice of Informal Patent Application (PTO-152)
- 6) ☐ Other: _____.

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1. The Election filed August 6, 2004 in response to the Office Action of July 8, 2004 is acknowledged and has been entered. Claims 1-2, 4-6, 19-16, 18-19, 25-30 are pending in the application and Claims 1-2, 4-6, 9-16, 18, 25-26, 30 have been withdrawn from further consideration by the examiner under 37 CFR 1.142(b) as being drawn to non-elected inventions. Claims 19, 27-29 are currently under prosecution.

2. It is noted that a review of the parent case of PCT/US00/16007 to which the instant application claims priority, US Application 09/329,917, did not reveal support for the instantly claimed invention, thus a priority date of June 9, 2000 has been established for the instantly claimed invention. If applicant disagrees with any rejection set forth in this office action based on examiner's establishment of a priority date of June 9, 2000 for the instantly claimed application serial number 10/009,861, applicant is invited to submit evidence pointing to the serial number, page and line where support can be found establishing an earlier priority date.

3. Applicant's election with traverse of Group I, claims 19 and 27-29 and the species paclitaxel is acknowledged. The traversal is on the ground(s) that the inventions have not been shown to be independent and there would be no serious burden in the search of all of the Groups. This is not found persuasive. MPEP 802.01 provides that restriction is proper between inventions which are independent or distinct. Here, the inventions of the various groups are distinct for the reasons set forth in the Paper mailed July 8, 2004. As to the question of burden of search, the literature search, particularly relevant in this art, is not coextensive. Different searches and issues are involved in the examination of each group. For these reasons the restriction requirement is deemed to be proper and is therefore made FINAL.

Specification

4. The specification on page 1 should be amended to reflect the status of the parent application.
5. Although the specification has a section for the Brief Description of the Figures and Figures 9A-9F are mentioned, there is no description of these figures. Appropriate correction is required.

Claim Rejections - 35 USC § 112

6. The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.
7. Claims 19 and 27-29 are rejected under 35 USC 112, first paragraph because while being enabling for a method of treating prostate cancer in a subject in need of such treatment wherein said prostate cancer overexpresses HER-2/neu comprising the step of administering a humanized antibody against the extracellular domain of HER-2/neu or a human monoclonal antibody against the extracellular domain of HER-2/neu, does not reasonably provide enablement for a method of treating prostate cancer in an individual in need of such treatment comprising the step of administering an antibody to HER-2/neu. The specification does not enable any person skilled in the art to which it pertains or with which it is most nearly connected to make and use the invention commensurate in scope with these claims.

The claims are drawn to a method of treating prostate cancer in a subject in need of such treatment comprising the step of administering an antibody to HER-2/neu. This includes treating with (1) any antibody to HER-2/neu regardless of where it binds on HER-2/neu, (2) regardless of whether it cross reacts with other antigens including EDGF receptor, (3) regardless of whether the antibody is monoclonal or polyclonal, (4) regardless of whether the antibody is humanized for treatment of a human subject as contemplated, (5) regardless of whether or not the cancer cells express HER-2/neu (6) regardless of the extent of expression of HER-2/neu.

The specification teaches the immunohistochemically identified membrane overexpression of HER-2/neu in primary prostate cancer samples, see pages 87-96.

One cannot extrapolate the teaching of the specification to the scope of the claims. Although Agus et al, (Cancer Research, 1999, 59:4761-4764, IDS item), whose authors include the instant inventors, teach a successful method of treating prostate cancer in a subject comprising administering to the subject a therapeutically effective amount of Herceptin (see abstract) which is a humanized monoclonal antibody against the extracellular domain of HER-3/neu and Pegram et al (J. Clin. Oncol., 1998, 16:2659-2671) teach a successful method of treating a different epithelial cancer in a subject comprising administering to the subject a therapeutically effective amount of rhuMab HER2 (see abstract) which is also a humanized monoclonal antibody against the extracellular domain of HER-3/neu, neither the art of record nor specification teach any anti-HER-2/neu antibody other than a humanized monoclonal antibody that binds specifically to the extracellular domain of HER-2/neu that is effective as a treatment for HER-2/neu overexpressing tumors. In particular, Agus et al teach that Herceptin has a

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cytostatic growth-inhibitory effect on breast cancer cells overexpressing Her-2/neu and has demonstrated clinical activity in breast cancer patients whose tumors overexpress Her-2/neu (p. 4761, col 2) and Pegram et al teach the efficacy of rhMAB HER2 in clinical trials.

One cannot extrapolate the teaching of the specification to the scope of the claims because other than teaching that a limitation of the claimed invention includes treating prostate cancer in a subject with an anti-HER-2/neu antibody, and other than the prior art teaching of the successful treatment of a subject with prostate cancer with HERCEPTIN, neither the specification nor the art of record teach how to make a therapeutic antibody with the properties required for treatment of HER-2/neu overexpressing tumors so that they will function as claimed. In particular one would not expect to be able to practice the claimed invention with an antibody that was not specific for the extracellular domain of HER-2/neu, for example an antibody to the intracellular domain or an antibody that binds only to denatured HER-2/neu, because the antibody would not bind to malignant cells expressing ErbB-2, since the antibody could not contact the intracellular domain of the protein, would not be able to bind to an unfolded protein and therefore would not inhibit the cells growth and/or proliferation. The specification provides insufficient guidance with regard to these issues and provides no working examples which would provide guidance to one skilled in the art and no evidence has been provided which would allow one of skill in the art to predict that the broadly claimed method will function as claimed with a reasonable expectation of success.

As drawn to cross reactivity of the broadly claimed antibody. It is well known in the art, as taught by Karunakaran et al (EMBO J., 1996, 15:254-264) and

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Graus-Porta et al (EMBO J., 1997, 16:1647-1655), that HER-2/neu is a member of the EGFR family and shares homology with other members of the family. Given the shared homology it would be expected that antibodies that are not selective for HER-2/neu would cross react with, and be sequestered by, other members of the EGFR family. In particular it is known that anti-tumor antibodies must accomplish several tasks to be effective. They must be delivered into the circulation that supplies the tumor and interact at the proper site of action and must do so at a sufficient concentration and for a sufficient period of time. Also, the target cell must not have an alternate means of survival despite action at the proper site for the anti-tumor antibody. In addition variables such as biological stability, half-life or clearance from the blood are important parameters in achieving successful therapy. The antibody may be inactivated *in vivo* before producing a sufficient effect, for example, by degradation, immunological activation or due to an inherently short half life of the antibody. In addition, the antibody may not otherwise reach the target because of its inability to penetrate tissues or cells where its activity is to be exerted, may be absorbed by fluids, cells and tissues where the antibody has no effect, circulation into the target area may be insufficient to carry the antibody and a large enough local concentration may not be established. The specification provides insufficient guidance with regard to these issues and provides no working examples which would provide guidance to one skilled in the art and no evidence has been provided which would allow one of skill in the art to predict that the broadly claimed method will function as claimed with a reasonable expectation of success.

As drawn to monoclonal as opposed to polyclonal antibodies. The claims as written read on not only monoclonal but also polyclonal antibodies. As set forth

above, given the identity of HER-2/neu with other members of the EGFR family, it would be expected that a large majority of polyclonal antibodies would bind to epitopes that are shared among members of the EGFR family. These sequestered antibodies would not be available to treat the cancer and it could not be predicted, for the reasons set forth above that the broadly claimed method will function as claimed with a reasonable expectation of success using polyclonal antibodies.

As drawn to non-humanized antibodies for treatment of human subjects which is clearly contemplated by the specification, Winter et al (TIPS, 1993, 14:139-143) specifically teach that a major problem with the use of murine monoclonal antibodies in the treatment of human subjects is the development of human antimouse antibodies (HAMA) that can inactivate the injected antibodies. Thus, it would be expected that the injection of cross species antibody would result in anti-other species antibodies and/or cytotoxic T cells against the injected antibody. Further, Baselga et al (J. Clin. Oncol, 1996, 14:737-744) specifically teach that murine antibodies are limited clinically because they are immunogenic. To facilitate clinical investigations, Mab 4D5 (the murine parent antibody of HERCEPTIN) was humanized. The humanization resulted in a safe treatment which has dose dependent pharmacokinetics in phase I clinical trials (p. 737, col 2). Given the teaching in the art, it could not be predicted and it would not be expected that non-humanized antibodies would function as claimed, that is as a therapeutic for the treatment of prostate cancer in the human subjects.

As drawn to treatment of prostate cancers that do not overexpress HER-2/neu, US Patent No. 6,156,321 specifically teaches that among the drawbacks of antibody anti-tumor therapy is that antigen negative cells can survive and repopulate a tumor (col 1, line 64, col 2, line 2). Further Lewis et al (Cancer

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Immunology Immunotherapy, 1992, 37:255-263) specifically teach, in Table 2 in *in vitro* studies, that while proliferation of cell lines that over-express ErbB2 was inhibited by treatment with anti-ErbB2 antibodies, proliferation of cell lines that do not over-express ErbB2 was generally unaffected (page 259). Thus, no one of skill in the art would believe that it would be more likely than not that the invention would function as claimed in a prostate cancer that does not overexpress HER-2/neu. The specification provides insufficient guidance with regard to these issues and provides no working examples which would provide guidance to one skilled in the art and no evidence has been provided which would allow one of skill in the art to predict that the broadly claimed method will function as claimed with a reasonable expectation of success.

As drawn to treatment of prostate cancer regardless of the extent of expression of HER-2/neu, Pegram et al (J. Clin. Oncol., 1998, 16:2659-2671) specifically teach that Mab 4D5 and HERCEPTIN are known to have antiproliferative activity only against HER-2/neu-overexpressing human breast carcinoma cells *in vitro* and against *in vivo* animal models of breast cancer xenografts with HER-2/neu overexpression *in vivo* (para bridging pgs 2659-2660). Thus it would not be expected and could not be predicted that the successful HERCEPTIN therapy could be used for the treatment of prostate cancers that did not overexpress HER-2/neu. The specification provides insufficient guidance with regard to these issues and provides no working examples which would provide guidance to one skilled in the art and no evidence has been provided which would allow one of skill in the art to predict that the broadly claimed method will function as claimed with a reasonable expectation of success.

Claim Rejections - 35 USC § 102

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8. The following is a quotation of the appropriate paragraphs of 35 U.S.C. § 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless --

(a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.

9. Claims 19 and 27-29 are rejected under 35 U.S.C. § 102(a) as being anticipated by Agus et al, *Supra*.

The claims are drawn to a method of treating prostate cancer in a subject comprising administering to the subject a therapeutically effective amount of anti-Her-2/neu antibody to the subject (claim 19) the method further comprising administering an antitumor chemotherapeutic agent (claim 27), paclitaxel (claim 28), wherein the prostate cancer is androgen-dependent (claim 29).

Agus et al teach a successful method of treating prostate cancer in a subject comprising administering to the subject a therapeutically effective amount of Herceptin, an anti-Her-2/neu antibody to the subject (abstract) the method further comprising administering an antitumor chemotherapeutic agent (see abstract), paclitaxel (see abstract), wherein the prostate cancer is androgen-dependent (see abstract). All of the limitations of the claims are met.

Claim Rejections - 35 USC § 103

10. The following is a quotation of 35 U.S.C. § 103 which forms the basis for all obviousness rejections set forth in this Office action:

A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art

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are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

Subject matter developed by another person, which qualifies as prior art only under subsection (f) or (g) of section 102 of this title, shall not preclude patentability under this section where the subject matter and the claimed invention were, at the time the invention was made, owned by the same person or subject to an obligation of assignment to the same person.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. § 103, the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 C.F.R. § 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of potential 35 U.S.C. § 102(f) or (g) prior art under 35 U.S.C. § 103.

11. Claims 19 and 29 are rejected under 35 U.S.C. § 103 as being unpatentable over Baselga et al (J. Clin. Oncol., 1996, 14:737-744) in view of Okumura et al (Breast Cancer, 1997, 4(4):269-272), Myers et al (J. Natl Can Inst., 1994, 86:15, 1140-1145) Arai et al (The Prostate, 1997, 30:195-201, IDS item), Craft et al (Nature Medicine, 1999, 5:280-285, IDS item) and Zhau et al (Mol. Carcinogenesis, 1992, 5:320-327, IDS item).

The claims are drawn to a method of treating prostate cancer in a subject comprising administering to the subject a therapeutically effective amount of anti-Her-2/neu antibody to the subject (claim 19), wherein the prostate cancer is androgen-dependent (claim 29).

Baselga et al teach that HERCEPTIN is well tolerated and clinically active in patients with HER-2 overexpressing metastatic breast cancers (see abstract).

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Previous studies have demonstrated that the parent antibody, 4D5 is a potent inhibitor of growth, *in vitro*, and in xenograft models of human breast cancer cells that overexpress HER2 (p. 737, col 20).

Baselga et al teach as set forth, but do not teach a method of treating prostate cancer with HERCEPTIN.

Okumura et al teach the successful *in vivo* treatment of c-erbB-2 overexpressing human gastric carcinoma with HERCEPTIN (see abstract)

Myers et al teach that p185^{erbB-2} is expressed on cell membranes of epithelial neoplastic lesions, from which prostatic adenocarcinoma appears to evolve, and on cell membranes of localized and metastatic adenoacarcinomas (p. 1144, col 1). Further, the frequently strong expression of p185^{erbB-2} in both primary prostatic adenocarcinomas as well as matched nodal metastases from patients with stage D adenocarcinoma suggests that p185^{erbB-2} may be used as a potential target in novel therapies (p. 1144, col 2).

Arai et al teach that approximately one third of clinically localized prostate cancers express c-erbB-2 and that Myers et al, *Supra*, showed that increased expression of c-erbB-2 represents an early event in the development and progression of prostate cancer (p. 198, col 2).

Craft et al teach that prostate cancer progresses from a hormone-sensitive, androgen-dependent stage to a hormone refractor, androgen-independent tumor (see abstract) and that radical prostatectomy samples rarely contain androgen-independent disease and report varying frequencies of HER-2/neu overexpression (p. 283, col 2) and that most groups have focused on radical prostatectomy samples (p. 283, col 1).

Zhau et al specifically teach the observation of immunohistochemical staining of c-erbB-2/neu primarily around the plasma membranes of prostatic cancer cells (see abstract).

It would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made to treat the subset of patients with hormone-dependent prostate cancer that overexpress membrane HER-2/neu with the HER-2/neu specific antibody HERCEPTIN of Baselga et al and Okumura et al because Arai et al, Myers et al both specifically teach that HER-2/neu is overexpressed on at least a subset of prostate cancers and because Myers et al specifically suggest that, given the identified frequently strong membrane expression of HER-2/neu in prostate cancer tumors, that HER-2/neu may be used as a target in novel therapies for prostate cancer and because Baselga et al specifically teach that HERCEPTIN is well tolerated and clinically active in patients with epithelial HER-2 overexpressing tumors and that both *in vivo* and *in vitro* studies of the parent antibody, 4D5, have shown that this antibody is a potent inhibitor of growth of cancer cells and Okumura also teaches the successful treatment of a different type of epithelial tumor overexpressing HER-2 with HERCEPTIN. One would have a reasonable expectation of success in treating the subset of patients with hormone-dependent prostate cancer that overexpress membrane HER-2/neu with the HER-2/neu specific antibody HERCEPTIN of Baselga et al and Okumura et al because prostate cancer is also an epithelial tumor that overexpresses HER-2/neu and Myers et al and Zhau et al specifically teach that expression of HER-2/neu is found at the cell membrane, thus it would be expected that the antibody would successfully target these tumor cells. Given the above, one would have had a reasonable expectation of success in treating prostate cancer with the known

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effective and well tolerated anti-HER-2/neu antibody. Further, it would have been *prima facie* obvious to one of ordinary skill in the art, and one would have been motivated, to specifically treat hormone-dependent prostate cancer in order to treat diagnosed cancer in an early stage at time of diagnosis because Craft et al specifically teach that prostate cancer progresses from a hormone-dependent stage to a hormone-independent stage and that samples from radical prostatectomy, generally done at time of diagnosis, rarely contain hormone-independent disease and because Myers et al teach that increased expression of c-erbB-2 represents an early event in the development and progression of prostate cancer and that this expression is frequently strong membrane expression and because Veltri et al teaches that assay of radical prostatectomy specimens revealed that 96 of 124 samples express HER-2/neu, thus one would expect to successfully treat hormone-dependent prostate cancer with Baselga's well tolerated and clinically active antibody against HER-2/neu in prostate cancer that frequently strongly expresses HER-2/neu on tumor cell membrane.

12. Claims 19 and 27-28 are rejected under 35 U.S.C. § 103 as being unpatentable over Baselga et al, J. Clin. Oncol *Supra*, Okumura et al *Supra* in view of Okumura et al *Supra*, Myers et al *Supra*, Arai et al *Supra*, Craft et al *Supra*, Zhau et al, *Supra*, above and further in view of Shaw et al (Methods and Findings in Exp. Clin. Pharmacol., 1998, 20(2);111-114).

The claims are drawn to a method of treating prostate cancer in a subject comprising administering to the subject a therapeutically effective amount of anti-Her-2/neu antibody to the subject (claim 19) the method further comprising administering an antitumor chemotherapeutic agent (claim 27), paclitaxel (claim 28).

The references of the prior art teach as set forth above but do not teach combination therapy comprising HERCEPTIN combined with paclitaxel.

Shaw et al teaches the successful treatment of subjects with prostate cancer with paclitaxel (see abstract).

It would have been *prima facie* obvious to include the paclitaxel of Shaw et al in the method of the combined prior art references because Shaw specifically teaches the successful treatment of subjects with prostate cancer with paclitaxel.

Since paclitaxel had been taught by the prior art to be effective in the treatment of prostate cancer and it would be expected, for the reasons set forth above that HERCEPTIN would successfully treat prostate cancer, the instant situation is amenable to the type of analysis set forth in In re Kerkhoven, 205 USPQ 1069 (CCPA 1980) wherein the court held that it is *prima facie* obvious to combine two compositions each of which is taught by the prior art to be useful for the same purpose in order to make a third composition that is to be used for the very same purpose since the idea of combining them flows logically from their having been individually taught in the prior art. Applying the same logic to the instant claims, given the teaching of the prior art of that paclitaxel is effective in treating prostate cancer, it would have been obvious to treat prostate cancer with both the HERCEPTIN of the combined prior art references and paclitaxel because the idea of doing so would have logically followed from their having been individually taught to be useful as cytotoxic agents for the same purpose, treating prostate cancer. One of ordinary skill in the art would have reasonably expected to treat prostate cancer with either or both of these agents since both the HERCEPTIN of the combined references and the paclitaxel of Shaw et al had been demonstrated to kill tumors.

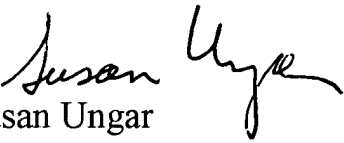
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13. No claims allowed.

14. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Susan Ungar, PhD whose telephone number is (703) 305-2181. The examiner can normally be reached on Monday through Friday from 7:30am to 4pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Jeffrey Siew, can be reached at (571) 571-0787. The fax phone number for this Art Unit is (703) 308-4242.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to the Group receptionist whose telephone number is (703) 308-0196.


Susan Ungar
Primary Patent Examiner
October 10, 2004